On page 11, line 14 through page 12, line 3, please replace with the following:

The URA blaster technique was used to construct a null mutant of C *albicans* that lacks expression of the Als1p. The *als1/als1* mutant was constructed in *C. albicans* strain CAI4 using a modification of the Ura-blaster methodology [W. A. Fonzi and M. Y. Irwin, *Genetics* **134**, 717 (1993)] as follows: Two separate *als1-hisG-IRA3-hisG-als1* constructs were utilized to disrupt the two different alleles of the gene. A 4.9 kb *ALS1* coding sequence was generated with high fidelity PCR (Boehringer Mannheim, Indianapolis, IN) using the primers: 5'-CCGCTCGAGATGCTTCAACAATTTACATTGTTA-3' (SEQ ID NO.1) and 5'-CCGCTCGAGTCACTAAATGAACAAGGACAATA3' (SEQ ID NO.2). Next, the PCR fragment was cloned into pGEM-T vector (Promega, Madison, WI), thus obtaining pGEM-T-*ALS1*. The *hisG-URA3-hisG* construct was released from pMG-7 by digestion with *KpnI* and *Hind3* and used to replace the portion of *ALS1* released by *Kpn1* and *Hind3* digestion of pGEM-T-*ALS1*. The final *als1-hisG-URA3-hisG-als1* construct was released from the plasmid by digestion with *Xhol* and used to disrupt the first allele of *ALS1* by transformation of strain CAI-4.

On page 13, line 5 through page 14, line 17, please replace with the following:

Referring to Figure 1, a comparison of the ALS1/ALS1 and als1/als1 strain showed that the ALS1 null mutant was 35% less adherent to endothelial cells than *C. albicans* CAI-12. To reduce background adherence, the adherence of the wild-type strain grown under non-ALS1 expressing conditions was compared with a mutant autonomously expressing A1s1p. This mutant was constructed by integrating a third copy of ALS1 under the control of the constitutive ADH1

promoter into the wild-type *C.* albicans. To achieve constitutive expression of the ALS1 in *C. albicans,* a blunt-ended PCR generated *URA3* gene is ligated into a blunt-edged Bg12 site of pOCUS-2 vector (Novagen, Madison, WI), yielding pOU-2. A 2.4 kb *Not1-Stul* fragment, which contained *C. albicans* alcohol dehydrogenase gene (ADH1) promoter and terminator (isolated from pLH-ADHpt, and kindly provided by A. Brown, Aberdeen, UK), was cloned into pOU-2 after digestion with *Not1* and *Stul.* The new plasmid, named pOAU-3 had only one *Bg12* site between the *ADH1* promoter and terminator. *ALS1* coding sequence flanked by *BamH1* restriction enzyme sites was generated by high fidelity PCR using pYF-5 as a template and the following primers: 5'-CGGGATCCAGATGCTTCA-ACAATTTACATTG-3' (SEQ ID NO.3) and 5'-CGGGATCCTCACTAAATGAACAAGGACAATA-3' (SEQ ID NO.4). This PCR fragment was digested with *BamH1* and then cloned into the compatible *Bg12* site of pOAU-3 to generate pAU-1. Finally, pAU-1 was linearized by *Xbal* prior to transforming *C. albicans* CAI-4. The site-directed integration was confirmed by Southern Blot analysis. Referring to Figure 1B, overexpressing *ALS1* in this P_{ADH1}-*ALS1* strain resulted in a 76% increase in adherence to endothelial cells, compared to the wild-type *C. albicans.* In comparing endothelial cell adherence of the wild-type to that of the overexpressing mutant, yeast cells were grown overnight in YPD at 25°C (non-inducing condition of Als1p). Als1p expression was not induced to reduce the background adherence of the wile-type, thus magnifying the role of Als1p in adherence through P_{ADH1}-*ALS1* hybrid gene. The adherence assay was carried out as described above. Statistical treatment was obtained by Wilcoxon rank sum test and corrected for multiple comparisons with the Bonferroni correction. $P<0.001$.

A monoclonal anti-Als1p murine IgG antibody was raised against a purified and truncated N-terminus of Als1p (amino acid #17 to #432) expressed using CLONTECH™ [Clontech] YEXpress

(™) yeast expression system (Palo Alto, CA). The adherence blocking capability of these monoclonal anti-Als1p antibodies was assessed by incubating *C. albicans* cells with either anti-Als1 antibodies or mouse IgG (Sigma, St. Louis, MO) at a 1:50 dilution. After which the yeast cells were used in the adherence assay as described above. Statistical treatment was obtained by Wilcoxon rank sum test and corrected for multiple comparisons with the Bonferroni correction. $P<0.001$. The results revealed that the adherence of the P_{ADH1}-ALS1 strain was reduced from 26.8%± 3.5% to 14.7%± 5.3%. Thus, the effects of ALS1 deletion and overexpression demonstrate that Als1p mediates adherence of *C. albicans* to endothelial cells.

On page 16, lines 5 - 14, please replace with the following:

If Efg1p stimulates the expression of *ALS1,* which in turn induces filamentation, the expression of *ALS1* in the *efg1/efg1* strain should restore filamentation. A functional allele of *ALS1* under the control of the *ADH1* promoter was integrated into the *efg1/efg1* strain. To investigate the possibility that *ALS1* gene product might complement the filamentation defect in *efg1* null mutant, an Ura *efg1* null mutant was transformed with linearized pAU-1. Ura^+ clones were selected and integration of the third copy of *ALS1* was confirmed with PCR using the primers: 5'-CCGTTTATACCATCCAAATC-3' (SEQ ID NO.5) and 5'-CTACATCCTCCAATGATATAAC-3' (SEQ ID NO.6). The resulting strain expressed *ALS1* autonomously and regained the ability to filament on Lee's agar. See Figures 4B and C. Therefore, Efg1p induces filamentation through activation of *ALS1* expression.

On page 18, line 20 through page 19, line 7, please replace with the following:

The fragment of *ALS1* was ligated into pQE32 to produce pINS5. In this plasmid, the protein is expressed under control of the *lac* promoter and it has a 6-hits tag fused to its N-terminus so that it can be affinity purified. We transformed *E. coli* with pINS5, grew it under inducing conditions (in the presence of IPTG), and then lysed the cells. The cell lysate was passed through a Ni^{2+} -agarose column to affinity purify the *ALS1*-6His fusion protein. This procedure yielded substantial amounts of *ALS1*-6His. The fusion protein was further purified by SDS-PAGE. The band containing the protein was excised from the gel so that polyclonal rabbit antiserum can be raised against it. It will be appreciated by one skilled in the art that the surface adhesin protein according to the invention may be prepared and purified by a variety of known processes without departing from the spirit of the present invention. The underlying polynucleotide sequence and the polypeptide sequence of Als1p are [is] listed in Figure 7 (SEQ ID NOS.7 and 8).